Chapter 4
Exposure Techniques: The Role of Extinction Learning

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4.1 Analogies Between Extinction Learning and Exposure-Based Therapies

There is now considerable agreement that exposure-based therapies represent one of the most effective treatment strategies for psychopathological conditions such as anxiety disorders (Craske et al., 2008; Hofmann, 2007; Hofmann, 2008; McNally, 2007) and drug abuse (Conklin & Tiffany, 2002). The historical roots of exposure-based therapies can be traced back to the studies by Watson and Rayner (1920) found in most textbooks concerning the history of psychology. In short, in their attempt to assess whether emotional responses could be experimentally manipulated in the laboratory, they presented "Little Albert" with a pet rat and allowed the child to play with it. Once Albert was familiar with the pet, they began to present a loud aversive noise each time Albert would reach to touch the rat. The intensity of the noise was high enough to elicit emotional reactions such as crying. After experiencing many rat–noise pairings, the child began to show the emotional reactions that were originally elicited by the loud noise but now to the rat, in the absence of the loud noise. This experiment was important at the time because it provided support for the view that some emotional reactions were learned and could be manipulated in the laboratory, placing psychology among those disciplines that exert experimental control over the phenomena under investigation. In addition, these experiments provided evidence that associative learning mechanisms can be responsible, at least in part, for the changes in behaviour that humans and other animals display in their natural environments. That is, the experience of two contiguous events (stimulus–outcome) results in the establishment of an association between them so that, upon subsequent encounters with one of the events (e.g., the stimulus), subjects will react to it based on the association that was established during those contiguous presentations. In other words, after experiencing two events in close temporal proximity, the stimulus predicts the occurrence of the outcome.

Humans, similar to other animals, are biologically prepared to take advantage of their prior experience to anticipate the occurrence of aversive events that are potentially life threatening (e.g., the appearance of a predator) and also events that promote survival, such as the encounter of food or a mate for reproduction. If the change in behaviour observed upon subsequent encounters with the one event depends on the organism's ability to associate (or link) these two events, experiencing the stimulus event in the absence of the outcome tends to restore the behaviour that was observed before any contiguous presentations was experienced. This change is called extinction (Pavlov, 1927). If one accepts that associations mediate our reactions to the stream of stimulation we experience in our daily lives, it provides a mean by which prior experiences, that evoke reactions of disproportionate magnitude in some anxiety disorders, can be brought under control in the therapist's office.

Exposure-based therapies exploit this idea by having clients experience, under the highly controlled environmental conditions of a therapeutic setting, events that may have been associated with threatening outcomes in the past but that are

no longer followed by these outcomes, which is by definition what we referred to as experimental extinction. Exposure therapies attempt to extinguish disproportionate responses that, in some cases, have seriously debilitating consequences in the lives of those who suffer clinical conditions such as anxiety disorders. It should be noted that exposure to stimuli in the absence of the outcome also applies to positive conditioned emotional reactions as is the case with drugs of abuse and palatable foods (i.e., obesity). In fact there is a large literature suggesting that stimuli predictive of drug administration or drug availability elicit cravings, which are a major cause of relapse after prolonged abstinence from drugs (Everitt & Robbins, 2005; Tiffany, 1990). The analogy between exposure therapies and extinction learning is obviously an oversimplification, as there are numerous factors that determine treatment success that go beyond the procedural and behavioural parallels.

Although exposure-based therapies are among the most successful ways to treat anxiety disorders and addiction, they are not immune to relapse, and in fact the conditions under which relapse is often observed strengthens the analogy between these exposure-based approaches and extinction learning. That is, clients relapse after some time has passed since treatment termination, and this obviously occurs outside of the therapist's office. Extinction learning, as it will be described later, is strongly dependent on the environment in which extinction and testing occur, and on the interval between extinction learning and testing. Thus, studies of experimental extinction that use animals in highly controlled settings are devoid of multiple confounds which are unavoidable when studying the effects of exposure in the therapist office such as high attrition rates and interpersonal characteristics of both the therapist and the client. This allows for a precise study of the conditions that increase the extent to which extinction learning will generalise to different environments and tolerate the passage of time, while permitting more secure conclusions and thus allow for theoretical developments.

In this chapter we will describe studies of experimental extinction conducted in human and non human animals, with the intention of highlighting: (1) the conditions under which extinction learning can be enhanced, as a means of translating knowledge obtained in highly controlled experiments to the clinical practise, and (2) some principles that underlie extinction learning and current associative explanations of extinction learning. Because in principle the potential of extinction to reduce the expression of prior experiences applies equally to negative (aversive) and positive (appetitive) outcomes, the studies described here are thought to apply equally to both sources of behavioural change. Although the goal of any psychological theory is to explain and predict the environmental conditions that cause changes in behaviour, one cannot deny the existence of neurobiological processes that underlie these changes. To put it differently, the brain mediates the changes in behaviour which we will assess in light of associative processes, and thus any attempt to separate these two is doomed to failure. Consistent with this rather obvious claim is the fact that exposure-based therapies are often administered in combination with pharmacotherapies (Hofmann, 2007; also see Chap. 6 in this volume).

4.2 Extinction Learning: Does It Erase the Original Memory, or Does It Create a New Memory?

One pressing issue in studies of extinction learning that has important consequences from a translational perspective such as that adopted here is whether extinction learning erases or destroys the original (i.e., excitatory) memory or, instead, results in the learning of a new relation between the stimulus and outcome (Stimulus → noOutcome) that interferes with the original memory (Dickinson, 1980). If extinction learning erases the original memory, successful extinction could remediate the detrimental effects of pervasive memories for ever, a goal that would place psychological therapy at the top of the chart in terms of its effectiveness. However, even in early studies of extinction learning, Pavlov (1927) and his colleagues documented that the change in behaviour brought by extinction learning was vulnerable to the passage of time, a phenomenon which Pavlov named spontaneous recovery. Logically, if recovery from extinction occurs in the absence of additional excitatory training (as is the case in spontaneous recovery), extinction learning cannot be accommodated by an explanation that assumes that it erases the original excitatory memory. That is, if the excitatory memory were to be destroyed by the extinction treatment, no recovery from extinction should be observed. Despite these early observations by Pavlov, some formal theories of learning have assumed that extinction can result in erasure (i.e., unlearning) of the original memory (Rescorla & Wagner, 1972), or at least in partial erasure (Stout & Miller, 2007). In controlled studies using animals, spontaneous recovery has sometimes been observed to be complete (Quirk, 2002). Even if some degree of unlearning occurs during extinction, convergent lines of evidence suggest that most of the original memory is not destroyed and these phenomena will be reviewed in Sect. 4.2.1.

Although in this chapter we will review evidence consistent with the idea that extinction does not result in unlearning or erasure of the original learning (Bouton & Bolles, 1979), erasure is often embraced by researchers interested in the neural and behavioural determinants of extinction learning (Quirk et al., 2010). Still, as noted by many authors (Bouton, Westbrook, Corcoran, & Maren, 2006; Bouton & Woods, 2008; Lovibond, 2004; Rescorla, 2004a), several observations lead to the conclusion that extinction learning does not erase the original learning. Following Bouton and Woods (2008), we will briefly describe six frequently cited recovery effects after extinction. To this list of recovery effects, we will add two more that also suggest that extinction does not erase the original learning (see Fig. 4.1).

4.2.1 Behavioural Phenomena Suggesting New Learning During Extinction

Evidence that extinction recovers with the passage of time, or *spontaneous recovery*, was documented by Pavlov and collaborators in their early studies on extinction

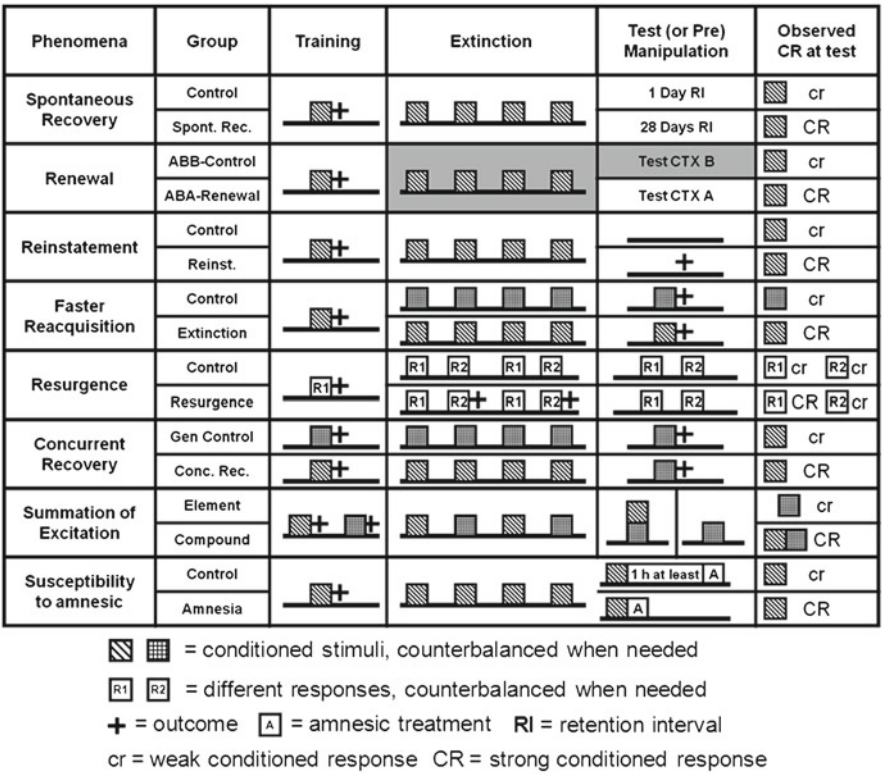

Fig. 4.1 Figure 4.1 depicts eight different phenomena which suggest that extinction does not lead to erasure of an association. See text for details of each procedure as well as sources of evidence. *Shaded boxes* mean that the procedure was conducted in a distinct environment

(Pavlov, 1927). Extinction learning is also vulnerable to changes in the context from where extinction learning occurred, and this has been called *renewal*. Renewal was observed first by Bouton and Bolles (1979), in an experiment in which rats first learned that an auditory stimulus was followed by a brief footshock in one context (A) and then experienced extinction training in which the auditory stimulus was no longer follower by footshock, but in a second context (B). The critical observation was that extinction (i.e., absence of responding) was observed when rats were tested in the extinction context (B) but fear to the auditory stimulus was strong when testing was conducted in the context where the auditory stimulus was followed by footshock (A). Thus, the notation ABA renewal (relative to ABB) will be used here and throughout this chapter to denote the context of training, extinction, and test, respectively. Renewal due to return to the context of original acquisition may be explained by residual excitation to the context summating with fear to the extinguished stimulus; in fact, this is a plausible explanation considering that the critical comparison between ABA and ABB renewal involves comparing stimuli tested in contexts that

differ in excitatory strength. However, extinction also recovers, although perhaps not as well, when the context of test is one that has no prior excitatory learning, as it is the case when testing is conducted in a third, neutral context, or in ABC renewal (Urcelay, Lipatova, & Miller, 2009). Thus, extinction learning seems to recover when testing is conducted in a context different than that of extinction learning. A third observation that joins spontaneous recovery and renewal in suggesting that extinction does not erase the original learning is *reinstatement* (Rescorla & Heth, 1975), which is the recovery from extinction learning observed after presenting the outcome alone (the unconditioned stimulus) after extinction and before testing. Reinstatement has important implications for clinical practise as seen in animal models of addiction where relapse after protracted abstinence is speeded with a small dose of the drug (Crombag, Bossert, Koya, & Shaham, 2008). Thus, recovery from extinction learning is better observed after the passage of time (e.g., spontaneous recovery), a change in the context where extinction learning occurred (renewal), or after the administration of a reminder achieved by the presentation of the consequence. Reinstatement is intriguing because it is context specific, when the outcome reminder is presented in a context different from that of testing; the reinstatement effect is largely attenuated.[1]

There are other ways to assess whether extinction erases the excitatory memory trace or instead produces new inhibitory-like learning that interferes with the excitatory content learned during initial acquisition. For example, if extinction erases the excitatory trace, retraining an extinguished memory should result in excitation at least similar to (but clearly never higher) to a second stimulus which has not undergone any prior excitation followed by extinction. In other words, if extinction erased the original memory, reacquisition should be similar for stimuli that have undergone acquisition followed by extinction and for stimuli that have undergone similar exposure but in the absence of excitatory learning (Delamater, 2004). Evidence supporting this prediction has been found in studies by Bouton (1986). The interpretation of these null results in terms of erasure is strengthened by observations that reacquisition after extinction sometimes proceeds slower than in a control group (Calton, Mitchell, & Schachtman, 1996; Denniston & Miller, 2003; Monfils, Cowansage, Klann, & LeDoux, 2009), a finding taken as consistent with a view of extinction that assumes inhibitory learning (Monfils et al., 2009).[2] With this said, the opposite finding has also been reported, namely *faster reacquisition* after extinction, a finding clearly at odds with an interpretation of extinction in terms of erasure (Bouton & Swartzentruber, 1989).

[1] In the drug-addiction literature, the term reinstatement has been adopted to refer to any recovery from extinction achieved by the presentation of an event that was present during drug self-administration, namely the drug itself (proper reinstatement) but also stimuli, contexts, and stress. See Crombag et al. (2008) for a revision of these findings.

[2] Dr MA Wood has made the interesting suggestion (Wood 2011; personal communication Jan 4) that the fact that reacquisition may be slower after extinction cannot be taken as evidence of erasure; because erasure should return the memory to a zero state after which reacquisition should proceed in the same way as a control group.

As noted by Bouton and Woods (2008), *resurgence* and *concurrent recovery* add to the list of phenomena suggesting that extinction memories are better understood in terms of new learning rather than erasure of excitatory traces. Resurgence has been primarily documented in instrumental learning and involves the use of two different levers that allow two different responses (R1 and R2) each followed by a pleasant outcome (i.e., a sweet food pellet). Critical is what happens after acquisition of R1, when this response is undergoing extinction, during which a second lever (R2) is concurrently reinforced (i.e., followed by the consequence). Resurgence, by definition, is the recovery from extinction seen to lever (R1) when the alternative lever (R2) is subsequently subject to extinction treatment (Winterbauer & Bouton, 2010). This phenomenon also has practical interest because often exposure-based therapies are administered concurrently with reinforcement of other behaviours. The effectiveness of the practise then depends, to some extent, on the other behaviours, which if extinguished will result in recovery from extinction in the target behaviour (R1).

Lastly, concurrent recovery refers to the observation that responding to an extinguished stimulus recovers when a second, unrelated stimulus, receives excitatory training (Weidemann & Kehoe, 2004). This is similar to resurgence at first glance, but actually the opposite in manipulation. In resurgence, recovery is observed when the alternative stimulus is undergoing extinction, rather than when the alternative stimulus is reinforced, yet both observations agree with the claim that extinction of the memory did not erase the memory trace.

The phenomena described above have become popular as criteria to determine whether a particular extinction treatment has erased a memory or not (Quirk et al., 2010). It should be noted however, that this interpretation is not impervious to logical problems. For example, evidence that there is no memory does not necessary indicate that the memory is not there. It could simply be the case that the memory is stored but not retrieved (Miller & Matzel, 1988). Thus, demonstrating memory erasure depends on proving the null hypothesis correct, that the erased memory in one group is not different than a group which has no memory (Nader & Hardt, 2009). Because there are ways to circumvent this problem which we will discuss below, it is worth pointing to additional phenomena which also suggest that extinction does not erase the original memory.

One such phenomenon suggesting that extinction leaves some of the original memory available was originally documented by Reberg (1972). In Reberg's study, animals received separate training of two stimuli (i.e., stimuli S1 and S2 which were never presented together) followed by separate extinction of each stimulus. One stimulus (S1) was extinguished to intermediate levels, whereas the remaining stimulus (S2) was extinguished until no conditioned responding was observed during three consecutive extinction trials. After extinction of S2 was complete, subjects received tests with each stimulus alone and with a compound of the two stimuli. If extinction erases the original memory (assuming unlearning is equivalent to erasure), tests with either stimulus alone or a compound should not make any difference. Contrary to this prediction, subjects showed strong conditioned responding when tested with the compound and weak responding when testing was conducted

with either stimulus alone. A second group of subjects received similar treatments as mentioned; however, S2 was additionally extinguished during 54 trials, during which no changes in responding were observed (extinction was already complete). During the tests in which S1 and S2 were presented separately, S1 evoked negligible levels of suppression, and S2 evoked behaviour that is consistent with S2 having acquired inhibitory properties during the extinction treatment. This pattern is consistent with the amount of extinction training that these two stimuli received. Still, strong conditioned responding was also observed in the second condition when both stimuli were tested together. This *summation of residual excitation* revealed by the compound test cannot be explained by an explanation that poses that extinction results in memory erasure. These results have been replicated by Rescorla (2006) who exploited this observation to test the predictions of a model that captures extinction as erasure (Rescorla & Wagner, 1972). All in all, the effect documented by Reberg adds to the above-mentioned list in suggesting that extinction, rather than erasing the original excitatory association, establishes new learning which is highly context dependent. Because testing the compound is clearly a different situation from experiencing each stimulus alone, recovery after testing the compound is not surprising.

Finally, another source of evidence for new learning during extinction is revealed by the vulnerability of extinction memories to amnesic treatments. The argument is that, if extinction memories are better captured as new learning, their consolidation ought to follow a similar time course as for other memories. In addition, their expression should be sensitive to reactivation manipulations known to have an effect on excitatory memories. A study by Briggs and Riccio (2007) recently showed that hypothermia-induced amnesia given soon after extinction of an inhibitory avoidance memory, but not 60 min later, attenuates the expression of the extinction memory. But time-dependent gradients should not be a criterion to establish that the memory represents new learning rather than unlearning, after all the process of unlearning could also need some time to get settled. Critically, Experiment 2 in their study showed that the amnestic effect of hypothermia on the extinction memory (which led to high levels of responding) could be alleviated if animals were cooled before testing, presumably because re-cooling them immediately before test reactivated the extinction memory that had presumably undergone amnesia. These experiments demonstrate that extinction memories, like excitatory memories (i.e., reinforcement), are susceptible to retrograde amnesia in a time-dependent fashion. In addition, the amnesic effect is sensitive to reactivation treatments, like new memories which need to undergo consolidation but also seem to recover with the appropriate reminder treatments (Misanin, Miller, & Lewis, 1968).

Despite Pavlov's early observation of spontaneous recovery and the wealth of phenomena suggesting that extinction does not erase the excitatory memory, the argument of memory erasure is frequently made, in particular in the last decade since the possibility of memory disruption after retrieval (i.e., reconsolidation) and new pharmacological treatments (see Chap. 6 by Hofmann et al. this volume) offer promising new avenues for therapists in the clinic whose goal is to relive their clients from the devastating consequences of traumatic events.

4.2.2 *Extinction as Context Dependent New Learning*

As an alternative to memory erasure of the excitatory association, it has been argued that extinction creates a new memory of the relationship between the Stimulus previously paired with the outcome, and the absence of the Outcome (S–noO), which depends heavily on the context for its expression. The context is defined as a collection of attributes given by distal features of the environment, but following Bouton (1993) this meaning is extended to temporal cues, so that the passage of time is understood as a change in temporal context. Put more precisely, time and space are equivalent and thus both changes of context should result in recovery from extinction. Although spatial and/or temporal contextual attributes modulate the expression of extinction memories, it is not clear which characteristics of extinction make it particularly susceptible to modulation by the context. One possibility, as discussed by Bouton, is that inhibitory S–noO memories are particularly context specific (Bouton, 1993). Alternatively, he also proposed that second learned memories about a particular stimulus are susceptible to modulation by the context. Because an extinction treatment is presumably inhibitory in nature and it is always administered after excitatory treatment, it is not possible to determine from extinction treatments alone which of these two criteria are necessary to observe modulation.

Sissons and Miller (2009) recently conducted experiments that assess these two alternatives. They administered excitatory training of one stimulus followed by inhibitory learning of that same stimulus, while also training a second stimulus that received similar training but in the opposite order. In other words, all subjects received excitatory and inhibitory training of two stimuli in two different stages, but the order was the opposite. They then tested subjects on different stimuli (whichever was trained last), but they did so for different groups at different intervals since the last phase of training. Subjects tested immediately after the end of the second phase of training responded much more to the stimulus that had received excitatory training last, relative to subjects that received inhibitory training last. This is consistent with the view that the second phase of training was dominant when these memories were tested immediately after. However, when different groups of animals were tested after a 21-day retention interval, responding was the opposite of that observed in the immediate test. Subjects responded more to the stimulus which received excitatory training first and inhibitory learning second (in other words, the dominance of inhibitory training seen immediately after the outset of stage 2 training was lost in favour or dominance of the first trained memory), and the opposite was true of subject that received training in the reverse order. These results suggest that there is nothing particularly special about inhibitory memories; it seems to be the case that second learned memories are particularly susceptible to modulation by the context.

Because clinical intervention such as exposure-based therapies are usually administered once the client has already acquired the fearful or appetitive relationship, it may be better to assume that the effects of the treatment, which is always

learned in a second stage, will invariably wane with the passage of time. In the next section, I will summarise some variables which, independently of the theoretical framework, enhance extinction learning and reduce recovery from extinction.

4.3 Manipulations that Enhance Extinction Learning

Assuming that extinction learning does not erase the original learning established before extinction takes place, it is worth pointing out variables that reduce recovery from extinction, because in practise these are the variables that may inform the clinician of alternatives to the traditional practise of exposure therapy with the objective of increasing its effectiveness. The summary presented here is not exhaustive and any interested reader may well consult additional literature on this issue (Laborda, McConnell, & Miller, 2011).

4.3.1 Massive Extinction

A strategy to reduce recovery after behavioural extinction has been to administer multiple extinction trials, assuming that more extinction trials will strengthen the extinction memory and alleviate recovery from extinction. For example, Tamai and Nakajima (2000), using fear conditioning in rats, administered training and extinction in the same context but tested in a different context, which should result in recovery from extinction (i.e., AAB renewal). Renewal was indeed observed after rats received 72 extinction trials, but not after 112 extinction trials. However, these parametric differences did not reduce ABA renewal in other groups, which typically results in robust recovery from extinction. These results suggest that extending extinction training does alleviate renewal, but only a weak form of renewal such as AAB renewal. Also using rats and fear conditioning, Denniston, Chang, and Miller (2003) administered 160 or 800 extinction trials. At issue was whether this extreme parametric variation would alleviate ABA renewal, and indeed they found that this was the case. Unfortunately other studies did not succeed in reducing renewal after massive extinction (Rauhut, Thomas, & Ayres, 2001), but this may be due to insufficient extinction given that the maximum number of extinction trials was substantially lower (100 and 144) than those administered by Denniston and colleagues. Further, the length of exposure therapy (i.e., number of sessions) has been directly assessed in several human studies conducted by Foa and colleagues. In general, these studies have found increased efficacy after prolonged exposure therapy relative to adequate controls (Foa et al., 2005). A recent meta-analysis, however, revealed no benefit of prolonged exposure therapy relative to other active treatments (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). It is possible that, through different mechanisms, different treatments may achieve similar beneficial outcomes, which should not undermine the potential of prolonged exposure to optimise extinction

learning and improve the effectiveness of exposure therapy. Overall, extending exposure seems to be beneficial in reducing recovery, although it is not entirely clear yet how much extinction should be given before treatment is terminated. The answer to this question may well depend on the strength of the fear memory, which makes it difficult but not impossible to determine how much exposure is necessary and sufficient to reduce relapse.

4.3.2 Extinction in the Presence of an Excitor

A candidate manipulation to boost extinction learning and reduce recovery is to increase the amount of fear during extinction trials, although this manipulation may not be the most pleasant for clients. Nevertheless, for extinction of fear to be successful, subjects do need to revaluate the original meaning of the fearful memory, and thus increasing the amount of fear during extinction may facilitate this revaluation. Indeed this outcome is predicted by some theories of learning (Miller & Matzel, 1988; Rescorla & Wagner, 1972), and is consistent with accounts of extinction that suggest that the amount of extinction learning is proportional to the strength of the conditioned response during extinction (Rescorla, 2001). This "rule of thumb" proposed by Rescorla is a powerful principle to anticipate the degree of extinction, and consistent with this idea, several studies have found that conducting extinction learning with two excitatory stimuli presented simultaneously, does alleviate several forms of recovery from extinction (Rescorla, 2000; Rescorla, 2006; Thomas & Ayres, 2004). However, this prediction is not entirely consistent with all theories, since configural models of learning anticipate less extinction when a cue is extinguished in the presence of a second excitatory cue (Pearce, 1987, 1994, 2002). These theories make this prediction because of their emphasis on configural processes occurring when two or more stimuli are presented simultaneously during extinction. Thus, configural theories anticipate recovery from extinction when it is conducted with two stimuli because they posit that during test the presentation of only one stimulus reduces transfer of extinction learning due to the change in stimulation occurring from extinction learning to test, a process that is called generalisation decrement. In fact, studies with pigeons in appetitive preparations have confirmed this prediction, namely that less extinction is sometimes observed when extinction is conducted in the presence of a second excitatory stimulus (Pearce & Wilson, 1991).

Although the reasons for these discrepancies are not entirely clear, these conflicting results may well indicate that multiple processes operate when extinction is conducted in the presence of a second excitatory stimulus. In support of this notion, studies have found no benefit of extinction with a second excitatory cue relative to control animals which received similar amounts of extinction of a cue alone (Urcelay, Lipatova, & Miller, 2009). These studies, in addition, consistently revealed decreased extinction learning in separate groups that received extinction of an excitatory cue but in the presence of a second stimulus which did not undergo

excitatory learning (i.e., extinction in the presence of a neutral stimulus). In other words, the mere addition of a second stimulus, independently of its excitatory value, decreased extinction even when testing was conducted in the same context in which extinction took place (ABB), an outcome consistent with configural models (Pearce, 1987, 1994, 2002). Based on these findings, it was hypothesised that extinction in the presence of a second excitor may provide some benefit for extinction learning, but this benefit may be masked by generalisation decrement occurring between extinction and the test. In accordance, Urcelay and colleagues were able to alleviate ABC renewal when they extinguished in compound stimuli of different modalities and durations, which presumably minimised subjects' configuring stimuli during extinction learning and thus facilitated the observation of extinction learning during the test.

Studies using fear conditioning in humans have also failed to support the idea that conducting extinction in the presence of multiple excitatory stimuli facilitates extinction (Lovibond, Davis, & O'Flaherty, 2000; Vervliet, Vansteenwegen, Hermans, & Eelen, 2007), and these failures have also been interpreted in terms of generalisation decrement diminishing any benefits of presenting multiple cues during extinction. An alternative to conducting extinction in the presence of a second excitatory cue is to present the aversive event (i.e., outcome) during extinction, which should increase fear levels to the context during extinction and facilitate extinction learning. A study in humans (Vervliet, Vansteenwegen, & Hermans, 2010) and one employing rats in a fear-conditioning preparation both showed attenuated renewal by presenting during extinction unsignalled presentations of the footshock outcome interspersed with extinction trials (Rauhut et al., 2001). Although this alternative has implications for theories of extinction, it is difficult to see in practise how this would be implemented in a therapeutic situation without raising ethical concerns (see Chap. 2 by Deacon in this volume).

4.3.3 Use of Retrieval Cues Associated with Extinction

One technique to reduce relapse after exposure-based therapies is provided by the use of different stimuli, or objects, that clients will associate with the calmness and interpersonal support of the therapeutic setting, which they can then take with themselves to aid the retrieval of the support and calmness that surrounds the therapeutic environment. In other words, introducing novel stimuli to the extinction session, as long as they do not provoke any unconditioned effects, may be beneficial because of the potential that these stimuli will have, outside of the therapist's office, to reduce fear in a novel situation. In fact some interventions make use of safety objects to aid long-term effectiveness of therapeutic programmes while at the same time reducing relapse.

Whether these cues provide any benefit can be studied under controlled laboratory situations. For example, Brooks and Bouton (1993) used an appetitive preparation in which rats responded to the illumination of a food-predictive light by

nose poking in the food magazine situated inside the conditioning box. Once training was stable, all animals were shifted to extinction. During extinction, some animals were presented with a stimulus four times at the beginning of each extinction session and on 75% of the extinction trials immediately before the cue. Thus, the stimulus clearly came to signal the extinction session and the extinction trials, perhaps functioning as an occasion setter (Holland, 1992), as the stimulus added during the extinction session did not have any excitatory or inhibitory properties on its own. Critically, when subjects were given a test six days later, those that had this neutral stimulus presented before the first test trial showed less spontaneous recovery. Presumably, the stimulus was capable of facilitating retrieval of the extinction session and consequently attenuated responding during a delayed test. These findings were soon replicated but in a counterconditioning design that differs from extinction in that during the second phase subjects are usually presented with the same stimulus they experienced during the original training, but now paired with a different consequence (Brooks & Bouton, 1994). Similar findings have been reported in humans. For example, Vervliet et al. (2007) administered electrodermal conditioning to a stimulus in humans, which then was followed by extinction training. However, before conditioning and extinction began, each phase was associated with a particular cue which signalled that the acquisition or extinction session had begun. When subjects were presented with the retrieval cues immediately before the test, they showed substantially more recovery from extinction when tested in the presence of the acquisition retrieval cue, although the extinction retrieval cue was not able to alleviate recovery from extinction.

Taken together, these findings in rats and humans suggest that retrieval cues present during acquisition or the extinction treatment can modulate the amount of recovery from extinction, and thus are critical when considering relapse. They do so, presumably, by virtue of their capacity to facilitate retrieval of memory of the extinction session which makes extinction (and counterconditioning) less vulnerable to the multiple sources of relapse that may be encountered on a day in the life of a patient.

4.3.4 Extinction in Multiple Contexts

Memories of extinction are best characterised as being highly context dependent and to transfer poorly to novel situations, and we just saw that stimuli that are associated with extinction can act like contexts and facilitate transfer of extinction between contexts. Similarly, it could be speculated that conducting extinction in multiple contexts will prevent the context specificity of extinction and facilitate its retrieval in new situations (Bouton, 1991). This prediction has important implications as many exposure-based treatments include exposure in situ and also in novel situations. Gunther, Denniston, and Miller (1998) conducted an experiment in which different groups of rats experienced fear conditioning followed by extinction either

in a single context or in three different contexts. The results indicated that extinction in multiple contexts did strengthen the extinction memory, as evidenced by less ABC renewal. However, a follow-up experiment suggested that this benefit was not observed if the excitatory memory was trained in multiple contexts, suggesting that subjects experiencing traumatic events in multiple locations would not benefit from extinction in multiple contexts.

In addition, other studies have suggested that the effect of extinction in multiple contexts is constrained by other experimental variables; thus, not all studies have replicated the original findings by Gunther and colleagues (Bouton, García-Gutiérrez, Zilski, & Moody, 2006; Neumann, Lipp, & Cory, 2007). Nevertheless, the beneficial effects of extinction in multiple contexts has been replicated in fear conditioning (Thomas, Vurbic, & Novak, 2009), in studies involving taste aversions (Chelonis, Calton, Hart, & Schachtman, 1999), and in humans (Neumann, 2008). Importantly, the benefits of extinction in multiple contexts upon recovery from extinction has been observed in spider-fearful participants, a finding in a critical population which suggests that this manipulation should be taken seriously due to its potential to alleviate return of fear and subsequent relapse (Rowe & Craske, 1998; Vansteenwegen et al., 2007).

4.3.5 Interval Between Acquisition and Extinction

Several studies have assessed whether extinction learning immediately after acquisition alleviates recovery from extinction. This variable is important because interventions such as immediate debriefing have been adopted as a strategy for dealing with traumatic events (Campfield & Hills, 2001), although the success of these manipulations has been questioned in a meta-analysis (van Emmerik, Kamphuis, Hulsbosch, & Emmelkamp, 2002). Studies in the laboratory using rats and pigeons in appetitive Pavlovian or instrumental preparations have found that when the interval between acquisition and extinction is lengthened, extinction is less (as opposed to more) vulnerable to different manipulations that induce recovery (Rescorla, 2004b). For example, rats received training in which two different cues were each followed by food pellets before one of them was extinguished completely. At the end of extinction of the first cue, all subjects experienced extinction of the alternative stimulus. This and other experiments consistently demonstrated that delayed extinction was resistant to spontaneous recovery assessed five days later, suggesting that delayed, rather than immediate extinction, is beneficial for extinction as it decreased its recovery. However, studies in rats using fear conditioning have observed that extinction given ten minutes after fear acquisition can alleviate recovery assessed through renewal, spontaneous recovery, and reinstatement (Myers, Ressler, & Davis, 2006). It should be noted that the data do not seem to reveal a large behavioural effect of immediate extinction, as if immediate exposure may only slightly dampen fear which does not show recovery. This becomes evident when immediate extinction is

compared with delayed exposure which substantially decreases fear but shows recovery given the appropriate treatment (Myers et al., 2006).

The reasons for these discrepancies are not clear, although soon after the publication of these latter findings, experiments run in several laboratories using rats and fear conditioning followed by extinction showed no benefit of immediate extinction when given up to 6 h after fear acquisition, compared with extinction given a day after fear acquisition (Archbold, Bouton, & Nader, 2010; Chang & Maren, 2009; Maren, Chang, & Thompson, 2006; Woods & Bouton, 2008). Although some studies have replicated the basic finding by Myers et al. (2006), the benefit of the immediate extinction treatment was better observed when testing was delayed rather than soon after extinction treatment (Johnson, Escobar, & Kimble, 2010). In general, fear-conditioning studies in humans have failed to observe benefits from immediate extinction (Alvarez, Johnson, & Grillon, 2007; Huff, Hernandez, Blanding, & LaBar, 2009; Schiller et al., 2008), although other studies have been able to replicate the immediate extinction effect but under select circumstances (Norrholm et al., 2008).

Lopez and colleagues (Lopez, de Vasconcelos, & Cassel, 2008) trained rats in a water maze (i.e., spatial memories) and administered extinction at much longer intervals after acquisition (5 days vs. 25 days). They found that delayed extinction treatment does not result in appreciable extinction when given on separate days. In fact, 25 days after acquisition, three exposure sessions given on three consecutive days resulted in a progressive improvement in spatial memory (Lopez et al., 2008; also see Rohrbaugh & Riccio, 1970). This difference presumably results from the use of spaced extinction sessions given on separate days, perhaps because (relatively few) spaced exposure given 25 days after acquisition, when the memory has been consolidated, may act as reminders rather than effectively extinguish the original memory. This is not surprising, as seen below; protocols employing largely spaced extinction trials may sometimes act as reminders rather than trigger behavioural extinction (Cain, Blouin, & Barad, 2003). Indeed, a follow-up study in which immediate (5 day) and delayed (25 day) extinction was given on three consecutive trials, but all in one day, found no differences between 5- or 25-day-old memories, presumably because the consecutive extinction trials prevented the repeated reactivation of the excitatory memory that competes with the establishment of extinction memory. Similar findings were observed when the physical salience of the cue was increased; suggesting that exposure to strong memory cues facilitated extinction learning in detriment of memory reactivation. Thus, these experiments in spatial learning suggest that the effectiveness of immediate vs. delayed extinction depends, at least in part, on the strength of the memory and the induction of behavioural extinction. When old memories are given a widely spaced extinction regimen, exposure increases the strength of the memories instead of behavioural extinction, a finding that has recently received support using fear preparations (Inda, Muravieva, & Alberini, 2011; Rohrbaugh & Riccio, 1970). These results highlight the complex nature of exposure treatments which not always result in behavioural extinction, in particular when given long after the memory has been acquired and with a widely spaced exposure regimen.

4.3.6 The Spacing of Extinction Trials

It is widely acknowledged that spaced acquisition is beneficial for memory retention (Barela, 1999). In the clinic, the amount of time between sessions may be an important variable and there is a large variability in the spacing of sessions between treatments. For example, some treatments involve massed exposure limited to a week or so, whereas conventional treatments are administered weekly over a period of several months. In addition, some have distinguished within-session reduction in fear from between-session reductions in fear (Craske et al., 2008). In the laboratory, the spacing of extinction trials has received a great deal of attention, but unfortunately it is not yet entirely clear which alternative (e.g., massed vs. spaced) is better. In some studies, massed extinction has been observed to enhance extinction (Cain et al., 2003; Rescorla & Durlach, 1987), the opposite finding has, however, also been observed (e.g., Urcelay, Wheeler, & Miller, 2009).

One explanation for these contradictory findings is revealed by the distinction made above between memory reactivation and successful extinction (Leet, Milton & Everitt, 2006). Extinction learning involves multiple presentations of the stimulus alone, which presumably activates a representation of the consequence and allows for an update of that memory representation, so that future encounters with the stimulus will be less likely to evoke a memory representation of the [aversive] outcome. If the memory is reactivated but not updated, the representation of the consequence evoked by the stimulus may actually be strengthened, as if the original relationship were being rehearsed and strengthened rather than learning a new relationship. Support for this speculation was found in a study by Lee and colleagues (Lee et al. 2006). Rats received fear conditioning training in which an auditory cue was consistently followed by a brief footshock. The following day, some animals experienced one presentation of the cue alone. One presentation of the stimulus was not sufficient to induce extinction, as evidenced by freezing to the stimulus tested on the day after the single presentation. However, it did seem to reactivate the memory making it vulnerable to disruption by an amnesic agent (Misanin et al., 1968). The implications for the spacing of extinction trials are that, when extinction trials are widely spaced, it is possible that each presentation of the stimulus reactivates a memory representation of the outcome without resulting in the formation of a new memory. Some empirical findings agree with this observation. In the studies by Cain et al. (2003) in which massed extinction was superior to spaced extinction in terms of diminished recovery, the subjects that received the spaced treatment did not stop freezing during the extinction session. In other words, these subjects never showed any extinction learning. It is not surprising then that these subjects showed strong fear during the delayed test (i.e., spontaneous recovery) or when tested in a different context (i.e., renewal). A somewhat similar distinction is that between the decrease in fear observed within a session and that observed between sessions (Craske et al., 2008; Davis, Ressler, Rothbaum, & Richardson, 2006; Drew, Yang, Ohyama, & Balsam, 2004). Within-session extinction reflects the update that occurs

on a given trial as a consequence of the learning that occurred on the trial immediately preceding that trial, whereas between session extinction (or transfer) reflects long-term changes as a consequence of prior learning. Moreover, the intervals between extinction trials (hereafter, intertrial interval [ITI]) within a session are spent in the same context, whereas the interval between sessions is spent on the home cages. This distinction is important in the context of the present discussion because rapid changes which are typically observed during the course of extinction may not necessary result in the enduring changes which are the main objective of therapy.

One candidate explanation for the discrepant findings observed in both humans and other animals is that massed extinction trials may increase within-session extinction, but this learning may transfer poorly to future encounters with the fearful stimulus. This conclusion is well captured by a human contingency learning study in which the ITI was manipulated in different groups of subjects (Orinstein, Urcelay, & Miller, 2010). After acquisition, subjects were assigned to one of three conditions. The Control condition received no extinction trials. A second condition named Spaced did experience extinction trials which were evenly distributed among presentations of several other stimuli that in this study acted as filler cues. A third Group, named Expanding (Bjork & Bjork, 2006), received extinction treatment similar in the number of trials to that received by Group Spaced, but in this group the distribution of extinction trials started being relatively massed and progressively became more spaced, resulting in longer intervals between extinction trials as extinction learning progressed. Stimuli were different foods that a fictitious character had eaten at a particular restaurant (i.e., the restaurant acted as a contextual stimulus), and the outcome was represented by adverse consequences of the food consumption (i.e., diarrhoea). Participants were required to rate each cue on each trial as it was presented, which allowed for the collection of data during extinction trials, and ultimately assess the effect of holding the ITI during extinction constant vs. increasing it as the extinction treatment progressed. To put it differently, subjects which received the extinction treatment with the expanding ITI started extinction with short intervals between extinction trials and progressively shifted towards longer ITI between extinction trials.

As predicted, subjects in the expanding condition showed a faster drop of their ratings than those receiving extinction trials with a constant ITI between extinction trials. The early benefit of massed extinction trials was eventually compensated by the amount of exposure, so that both groups ended the extinction session rating the target stimulus similarly. Intriguingly, a test of ABA renewal revealed no benefit of Expanding vs. Constant ITIs. Thus, the sharp decrease in ratings observed early during extinction did not attenuate recovery from extinction, consistent with the above-mentioned reviews highlighting the differences between what is observed during extinction, and transfers to situations outside the extinction setting. In fact, if one were to learn a lesson from these experiments and try to translate to the clinic, it would be that therapists should rely little on the fear assessments obtained in the therapist's office, as it may well give them a picture that changes drastically once

the client has left the office. Fortunately, multiple assessments over time, and in different scenarios, are already standard in clinical practise.

The effect of spacing or massing extinction trials thus may differ depending on whether one looks at what happens during extinction and what happens on a subsequent test. There are various studies in which the spacing of extinction trials was systematically manipulated and then tested outside of the extinction session. As stated, these results are inconsistent and no clear picture has emerged. I will argue here that this possibly results from the use of different intervals during extinction, which may comprise more than one process. The relationship between increasing the ITI during extinction and the degree to which extinction learning is resistant to recovery is not linear, so that increasing the ITI during extinction (i.e., spacing extinction trials) may optimise extinction learning and alleviate recovery from extinction, but not when extinction trials are too spaced. When extinction is conducted with parameters that do indeed lead to extinction, an extinction trial n capitalises on the previous extinction trial $n-1$. However, if trial n is too far removed in time from the previous trial (i.e., presented a day later), it no longer benefits from the extinction that occurred on $n-1$, and thus is less effective in producing extinction learning. For example, it has been observed that a single presentation of a previously trained stimulus will elicit conditioned responding, presumably because it brings the memory into an active state. But one trial alone does not necessary produce extinction learning; in fact a single presentation may reactivate the memory and strengthen it (Inda et al., 2011; Pavlov, 1927; Rohrbaugh & Riccio, 1970). If trial $n-1$ occurs long before trial n, trial n will be experienced as a reactivation trial, a reminder of the aversive situation.

Recent neurobiological evidence suggests that one single presentation of a cue starts a cascade of molecular events that are different from those observed after ten presentations of a similarly trained stimulus (Lee et al., 2006). In fact, some have argued that the molecular cascade responsible for extinction learning is not initiated until the presentation of the stimulus itself has ended (Pedreira, Pérez-Cuesta, & Maldonado, 2004). This is consistent with the conflicting results discussed above. In the report by Cain et al. (2003), rats that received the spaced extinction protocol did not decrease freezing during the extinction session; in other words, the behaviour in these animals never seemed to extinguish. The fact that these animals responded more during the tests of recovery from extinction than animals trained with massed extinction trials does not suggest that extinction is more effective with massed extinction training because, in the groups extinguished with spaced trials, extinction never happened in first place. Obviously the optimal interval between extinction trials will vary depending on the task being used, the response under consideration, and a myriad of different conditions such as the strength of the original memory, and the depth of processing during extinction. One principle seems to emerge from this discussion though. Spacing extinction trials strengthens extinction learning, but only to a certain extent, when extinction trials are too spaced, extinction learning no longer takes place. The argument is that spaced presentations of the stimulus no longer benefit from the immediately preceding trial.

4.3.7 Pre-Extinction Retrieval

Traditionally, memories have been thought to necessitate a period of time (at least 6 h) after training for them to become consolidated and permanently stored in the brain (McGaugh, 1966, 2000). If an amnestic treatment is given within this critical window, it will weaken the memory being stored. Although appealing due to its simplicity, this idea was challenged by experiments in which the amnestic treatment was given postmemory reactivation a day later, a long time after the putative critical window of consolidation (Misanin et al., 1968). According to consolidation theory (McGaugh, 1966), once a memory is stabilized it should no longer be vulnerable to the effect of the amnestic treatment. In the experiment by Misanin et al. (1968), the amnestic treatment was equally effective when given after memory reactivation, suggesting that reactivated memories could also be affected by the amnesic treatment. The idea that reactivated memories can undergo a second "round" of consolidation, or reconsolidation, regained popularity recently after studies in fear conditioning replicated these findings with a high degree of specificity in the neural substrates underlying the behavioural observation (Nader & Hardt, 2009; Nader, Schafe, & Le Doux, 2000).

The idea that once active, memories can be modified is not new (Lewis, 1979; Bjork, 1975). Nevertheless, the last decade has seen a vigorous re-emergence of studies investigating the mechanisms and neurobiological processes underlying consolidation and reconsolidation, as these treatments could potentially act in a similar way as extinction-like treatments, in the sense that they may allow targeting specific memories, reactivating them, and then attempting to decrease the strength of the memory traces. The administration of amnesic treatments ordinarily involves potentially toxic drugs which may have undesirable effects due to a lack of specificity; therefore, this is not currently a standard practise. Alternatively, after memory reactivation one could administer extinction treatment when the memory is active and hence hyper vulnerable to the effects of extinction, while at the same time controlling to some extent for specificity in terms of the content of the memory. If, after reactivation, the memory is in a labile state, then following reactivation with a robust extinction treatment may enhance extinction and alleviate recovery from extinction. This was the rationale used by Monfils and colleagues in a series of studies in rats using fear conditioning (Monfils et al., 2009). They trained rats in a fear-conditioning preparation and a day after they gave them a single extinction session with 20 extinction trials. All groups received extinction, but they differed in the length of the interval between the first extinction trial (which acts as a reminder and produces memory reactivation) and the rest of the extinction regimen. That is, different groups received extinction training 10 min, 1, 6, or 24 h after the first presentation, which presumably produced memory reactivation. Subjects that experienced extinction treatment within an hour after reactivation, but not 6 or 24 h after reactivation (or no reactivation, with an ITI of 3 min between extinction trials), showed attenuated renewal, spontaneous recovery, and reinstatement. In addition, these subjects were slower to reacquire a fear response to the extinguished cue.

The mechanism by which this occurs is far from fully understood in that the critical difference between subjects which received extinction 10 min after reactivation and those who did not, was that the first ITI between extinction trials 1 and 2 was only 7 min longer (the ITI during extinction was 3 min). This finding was soon replicated in human fear conditioning (Schiller et al., 2010), using similar parameters to those used with rats, and with the addition of a within-subjects design, thereby suggesting some generality to this finding. Moreover, in humans, the benefit of retrieval prior to extinction was observed in a test conducted 12 months after acquisition and extinction. Unfortunately, replications from other laboratories have not always been successful (Chan, Leung, Westbrook, & McNally, 2010; Soeter & Kindt, 2011). For example, Chan et al. (2010) found in six experiments that, if anything, the reactivation trial increased renewal and reinstatement. They observed that the similarity between the context of training and the context where retrieval was administered may have been one reason for the lack of replication (also see, Soeter & Kindt, 2011).

Whether reactivation (which is equivalent to a single extinction trial) prior to extinction truly facilitates unlearning-like learning (in contrast to an interfering inhibitory-like memory) may require the test of time and replication. In studies conducted in the Psychological Laboratory at the University of Cambridge (Wood, 2010), the effect has been observed consistently in rats. Thus, Wood asked, for example, whether reactivation prior to extinction results in inhibitory memories that pass summation and retardation tests of inhibition, two canonical tests of conditioned inhibition[3] (Rescorla, 1969). Although memories that underwent reactivation prior to extinction were slower to reacquire excitatory properties (retardation test), they did not seem to pass a summation test for inhibition (Wood, 2010). In addition, Wood also assessed the specificity of reactivation prior to extinction, by conducting the mentioned protocol but in addition assessing the impact on acquisition of fear to a novel stimulus, which had not undergone any previous training. Surprisingly, she observed that administering reactivation followed by extinction rendered a (i.e., different) novel cue retarded in acquisition with the same outcome, suggesting that reactivation prior to extinction of the cue may produce some of its effects by changing the properties of the footshock representation (i.e., the outcome). This may explain why training of a novel cue was also retarded. In addition, for reactivation prior to extinction to be effective, subjects need to be removed from the context during the interval between reactivation and the subsequent extinction trials, otherwise the benefit of reactivation was no longer observed. In other words, the effect of reactivation prior to extinction depended on subjects being removed from the experimental setting, presumably because the reactivated memory needs to be updated and this is prevented if subjects remain in the context where retrieval occurred (Wood, 2010).

[3] In the associative-learning literature, inhibition refers to the explicit preventative relation between a stimulus and the outcome, which is inferred when the putative inhibitor attenuates the response elicited by an excitatory cue that has been trained separately (summation test), in addition to the putative inhibitor showing retarded emergence of excitatory learning (retardation test).

One pressing question for an explanation in terms of reconsolidation relates to the specification of the mechanism underlying the phenomenon. For example, given acquisition and reactivation prior to extinction treatment, one could speculate that a new instance of reactivation should destabilize the extinction memory and facilitate acquisition of fear, since the second reactivation after extinction should retrieve the extinguished (dominant) memory and allow for faster reacquisition. This outcome was not observed in Woods' experiments, but she observed that when the second reactivation was followed by reinforcement (i.e., the outcome) it reinstated the excitatory properties of the stimulus, which is consistent with the idea that for reconsolidation to occur the reactivated memory needs to be updated, as proposed by Lee (2009). Finally, another report that replicated the effect using tests of spontaneous recovery and renewal, also showed that if extinction (with and without prior reactivation) is conducted seven days after acquisition, there is no effect or reactivation prior to retrieval (Clem & Huganir, 2010). Overall, the available data so far suggests that there may be instances in which reactivation prior to extinction does facilitate extinction and alleviate recovery, but many boundary conditions apply, which makes it, to date, difficult to translate these findings directly into a clinical setting.

4.4 Theoretical Implications

In order to facilitate a brief summary of the findings reviewed in this chapter, we will describe two general theoretical approaches aimed to address the characteristics of extinction memories. One, which we will refer to as "associative," focuses on quantifying the strength of the connection between a stimulus and the outcome and is less concerned with temporal variables such as the interval between training and extinction, the benefit of conducting extinction in multiple contexts, or the effect of reactivating the excitatory memory before extinction. A second family of theories, which we will call "mnemonic" is less explicit about the strength of the connection between stimulus and outcome (and also S–noO), and focus on the conditions that constrain or enhance the expression of that learning. This distinction is rather general and made for the purpose of clarifying the emphasis of one or the other explanatory constructs. Ultimately, it may well be the case that a theory that is intended to account for the full range of phenomena will necessitate both approaches (i.e., hybrid).

4.4.1 Associative Theories

Associative theories of learning provide a quantitative measure of the strength of the connection between stimulus and outcome, but assume different factors to be critical in the update of these connections. For example, the Rescorla–Wagner model

poses that during extinction learning, the excitatory connection between the stimulus and the outcome that was formed during acquisition will decrease until it reaches a value close to zero. Thus, extinction is reflected by a loss in associative strength between the stimulus and the outcome, which is equivalent to assuming memory erasure. Despite this failure of the theory to account for a key signature of extinction learning such as its recovery, the model has been an invaluable source of predictions concerning other phenomena, many of which have successfully been tested in the laboratory (Rescorla, 2000). Models conceived soon after the R–W model have avoided this shortcoming by assuming that, during extinction training, subjects form a new S–NoO association which influences behaviour in a way opposite to the influence of excitatory associations formed during training (Konorski, 1967; Pearce & Hall, 1980). Although these models do not fare any better than R–W when explaining recovery from extinction (i.e., in principle they do not anticipate recovery from extinction), by assuming that inhibitory S–noO associations generalise less easily to new situations than excitatory associations do (Spence, 1936), these models are able to account for some forms of recovery from extinction. Variations of these models have proliferated in the literature in the last decades, perhaps due to the interest on extinction itself, in addition to the potential of extinction to inform which variables may turn out to be critical in the clinic (Gershman, Blei, & Niv, 2010; Redish, Jensen, Johnson, & Kurth-Nelson, 2007).

Associative models, although not fully accurate when it comes to anticipating recovery from extinction, do make specific predictions when during extinction the stimulus being extinguished interacts with other stimuli, as it is the case when extinction is conducted in the presence of a second excitor, or even a conditioned inhibitor (Lovibond et al. 2009). One possible reason why these theories fare well in these scenarios is that most of these models were designed to account for interactions between stimuli, like, for example, overshadowing or blocking. Because these models were designed with these phenomena in mind, they do not anticipate that extinction will recover, but they correctly predict what the net result will be of extinguishing a stimulus in compound with other stimuli. In addition, these models make some specific assumptions about the role played by contextual stimuli. Importantly, they treat the context like any other discrete stimulus, so that the context can enter in competition with the stimulus being extinguished, rather than modulating the expression of extinction which is what can be safely concluded to be at least one putative role of the context from the evidence reviewed above. Consistent with this assumption about the context are data supporting specific predictions made by these models but only when extinction is conducted with massed trials, a situation that leaves little room for context-alone exposure and thus is more likely to engage the context as a competing stimulus (Urcelay & Miller, 2010; Urcelay, Witnauer, & Miller, in press). Thus, associative models accurately predict interactions between stimuli during extinction, but fail to explain recovery from extinction, a characteristic that, for the sake of any translational effort, is critical since the analogy between extinction and exposure-based therapies is mostly based on the fact that behaviour recovers when some aspect of the situation change between extinction and test. That is, clients that receive exposure-based

therapies relapse when they leave the therapist office (a situation analogous to renewal) or with the passage of time, a situation analogous to spontaneous recovery (Orinstein et al., 2010).

4.4.2 Mnemonic Theories

The second family of models that we described above, the mnemonic theories, do not always specify the quantitative aspects of the change in behaviour, but they make more accurate predictions regarding the conditions that ensure the expression (or its absence) in similar or novel environments (i.e., contexts). The root of these theories can be found in verbal learning experiments conducted in the middle of the last century, which gave rise to numerous models of memory interference (Spear, 1978). An important point should be noted here: extinction itself is a form of interference in which a stimulus, during an initial phase, has an excitatory relationship with the outcome (S–O), but when extinction learning begins, that relationship changes because the stimulus no longer is followed by the outcome (S–noO), which is essentially a two-phase memory interference design. The model proposed by Bouton (1993) explains very well some characteristics of latent inhibition, which is similar to extinction but with the order of the treatments reversed (S–noO first followed by S–O in a second phase). A similar important aspect of this model is the treatment of contextual information (also see; Spear, 1978). Mnemonic models assume that the context functions like facilitator of retrieval for extinction, and this is why they are so successful in anticipating the difficulties observed in the laboratory and in the clinic for exposure (i.e., extinction) to transfer to situations outside the context where extinction occurs. These models assume that the context modulates the expression of extinction, presumably because second learned information, which produces interference, is highly context dependent (Sissons & Miller, 2009). Phenomena like the benefit of reactivating memories before extinction (Monfils et al., 2009; Wood, 2010) are closer in spirit to these models than to associative models. The reason for this is that the phenomenon of reconsolidation is indeed related to interference much more than it relates with competition between different sources of information, in the sense that memories are assumed to be in different states, although there is little specification of the quantitative attributes of these memories. Rather, explanatory constructs such as reconsolidation provide descriptions of the underlying processes responsible for the phenomena under question (i.e., memory reactivation, which then becomes context dependent (DeVietti & Holliday, 1972) just like extinction treatments and exposure-based therapies).

Taken all together, associative and mnemonic theories of extinction seem to emphasise different aspects of extinction learning. Whereas associative models concentrate on what occurs during extinction learning (hence their accurate predictions regarding interactions between stimuli during extinction), mnemonic models fare much better with the expression of extinction, a stage relatively isolated from the process of extinction on itself (Urcelay & Miller, 2008). Whether the critical

differences between these families of theories arise from their differential treatment of contextual information is not entirely clear, although recent fear-conditioning experiments conducted in rats suggest that seemingly trivial parametric variations may result in contexts playing largely different roles in information processing (Urcelay & Miller, 2010; Urcelay et al., in press). This may give some insight concerning the successes and failures of these models, as they make largely different assumptions about the function played by contexts. A challenging possibility is to incorporate these different functions of contextual information into one parsimonious model. These two functions, it should be noted, are not mutually exclusive; it is likely that contexts can play both roles at once. If this is done properly, then a theory that can specify the circumstances under which contexts will behave like any other stimulus or instead modulate the expression of stimuli trained inside them will likely provide a full account of the phenomena related to extinction learning, and perhaps better approximate the needs of those working in the clinical setting.

Overall, in this chapter we have characterised current understanding of extinction learning, which seems to be best captured as new learning of the relationship between stimuli and outcomes (indeed S–noO), rather than erasure of previously learned relationships. We further described several strategies that have been developed in the laboratory with the intention of overcoming what seems to be a critical characteristic of extinction learning, which is its recovery. Of course, these strategies are not recipes but rather, as should be obvious to the reader, avenues that are being explored in the laboratory and are still subject to much heated debate. Finally, we have outlined some of the conflicts between families of models aimed at explaining extinction learning, with the intention of highlighting those areas in which integration is needed. Together with pharmacotherapies, behavioural approximations to anxiety disorders and addiction currently represent the first line of treatment, and extinction processes seem to have an important role in their effectiveness. A challenge for the future is to better understand these, and perhaps their interaction (see Chap. 6 by Hofmann et al. this volume), to better achieve the desired clinical outcome that ultimately will lead to maximal success.

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